The Effects of Western Diet on Hippocampal Dependent Cognitive Function in a College-Aged Population

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This paper reviews the relevant literature and proposes an experimental approach to assessing the relationship between consumption of a western diet and cognitive functioning for young adults in the collegiate environment. Previous research has established a relationship between the western diet and obesity. The western diet is characterized as an energy-dense diet that is high in fat, particularly saturated fat, and processed sugars and low in unrefined carbohydrates. Further research has linked the consumption of the western diet to impaired cognitive function in animal and human models. While studies have shown that cognitive function is impaired by the western diet, the underlying mechanism that produces poor performance on cognitive tasks and promotes obesity has not been determined. One model proposes that an impaired ability to inhibit competing memories during memory recall produces poor performance on cognitive tests. Furthermore, the inability to inhibit memories of positive digestive outcomes may lead to overconsumption of the western diet and contribute to obesity. The present paper describes an experimental approach that could investigate this theory by evaluating college student's executive cognitive function and their ability to inhibit memories while also tracking their eating habits and weight gain. In addition to cognitive tests, this study would track the participant's academic performance to determine if cognitive impairment has a functional impact on academic work. Further research in this area is important as it may lead to new early intervention methods aimed at preventing cognitive decline and would add to the current dearth of knowledge about how diet, obesity, and cognition are related in young adults.

INTRODUCTION

The United States has the highest obesity rates in the world according to and the Organization for Economic Co-Operation and Development reports (Sassi & Devaux, 2012). The negative emotional, social, and most importantly health consequences of obesity have been widely documented (Puhl & Brownell, 2006; Kirk, Penney & McHugh, 2009).

In regard to health, obesity has been linked to serious chronic diseases including type 2 diabetes, hypertension, various cancers, coronary artery disease and stroke (Kirk, Penney & McHugh, 2009). The World Health Organization (WHO) defines overweight and obesity as "abnormal or excessive fat accumulation that may impair health" (World Health Organization, 2013). Body mass index (BMI), is a ratio of weight-for-height in adults that is used to classify overweight and obesity. WHO defines a BMI greater than or equal to 25 as overweight and a BMI greater than or equal to 30 as obese, which will be used as standard throughout this paper unless indicated otherwise.

Human studies have associated increased BMI with a number of negative effects on the brain and cognitive functioning. These associations include reduced gray matter (Soreca, Rosano, Jennings, Sheu, Kuller, Matthews, Aizenstein & Gianaros, 2009), reduced white matter integrity (Stanek, Grieve, Brickman, Korgaonkar, Paul, Cohen & Gunstad, 2011) deficits in executive functioning (Smith, Hay, Campbell & Trollor, 2011), global cognitive function, semantic memory, psychomotor speed (Eskelinen, Ngandu, Helkala, Tuomilehto, Nissinen, Soininen & Kivipelto, 2008) prospective memory, delayed verbal memory, reduced synaptic

activity, and inflammation (Kanoski & Davidson, 2011). Socially, studies have shown that obese people may be denied jobs and promotions more frequently than normal weight people, may be treated more poorly by coworkers and employees, are more frequently ridiculed by peers and may be viewed negatively by educators (Puhl & Brownell, 2006).

Despite these consequences, obesity rates have continued to increase at an alarming rate and data has clearly shown that this trend is expected to continue (Wang, McPherson, Marsh, Gortmaker & Brown, 2011). The fact that many people who are obese are not able to return to a normal weight, combined with the fact that people continue to become obese knowing many of the negative consequences of obesity, implies that an underlying mediator for obesity may be present in the modern western world.

The question facing researchers is not *if* people are becoming obese, it has already been clearly established and documented that obesity rates are increasing. The question facing researchers is not even *how* people are becoming obese. The obesogenic environment and the western diet have been clearly linked to obesity across populations. The question facing researchers is what is the underlying mechanism that causes some people to become obese while others are not similarly affected by the western diet.

This paper will look at young adulthood and the collegiate environment as an important potential area of study in western diet and cognitive functioning research. Further research in this area is important as it may lead to new early intervention methods and information on the progression of cognitive decline. The paper will

highlight studies that link the western diet to obesity, and studies that have linked the western diet to cognitive decline. These studies have show that the western diet negatively affects a variety of cognitive functions in both animal and human models. Cognitive effects range in severity from barely noticeable memory impairment as seen through academic performance, to Mild Cognitive Impairment to Alzheimer's disease (Petersen, Stevens, Ganguli, Tangalos, Cummings & DeKosky, 2001)

OBESITY IN THE UNITED STATES

Obesity has become a major concern in the United States. In the past twenty-three years, obesity in the US has more than doubled. In 1990, the obesity average in the US reported by the CDC was 12% (Ogden, Carroll, Kit & Flegal, 2012). In 2010, the CDC reported that this average had risen to 33.8% of adults and almost 17% of children and adolescents. The CDC defines obesity for adults as a Body Mass Index (BMI) greater than or equal to 30 and obesity for children as a BMI greater than or equal to the age and sex specific 95^{th} percentiles of the CDC growth charts. Recognizing the significant increase in obesity from 1990 to 2010, Wang et al used historic data derived from the National Health and Nutrition Examination Survey to predict that by 2030, roughly 50% of the US population will be obese (Wang, McPherson, Marsh, Gortmaker & Brown, 2011). With such rapid growth in the number of obese individuals in the US, the study of the causes of obesity has become increasingly important.

The type of environment that promotes obesity is referred to as an "obesogenic" environment (Kirk, Penney & McHugh, 2009). The main component of an obesogenic environment is the consumption of energy-dense diets that are high

in fat, particularly saturated fat, processed sugars and low in unrefined carbohydrates. The obesogenic environment also includes a lack of energy expenditure deriving from a sedentary life style, though to a lesser degree. While genes have been implicated as a contributing factor to obesity, with estimates for heritability of obesity ranging from 30-70% (Martinez-Hernandez et al. "Genetics of Obesity"), genetics alone cannot explain the rapid increase in obesity seen at the national level (Kirk, Penney & McHugh, 2009). It seems as if genes that promote deposition of adipose fat and therefore overweight and obesity, are expressed where the environment allows and encourages their expression (Lobstein, Baur & Uauy, 2004).

The college environment is a microcosm of an obesogenic environment, providing greater independence over food choice and the opportunity for students to become more sedentary with no required physical education[1]. Hajhosseini et al. found that while there was no statistically significant change in total energy intake in a study of college freshman weight gain, there were significant increases in total carbohydrate intake (Hajhosseini, Holmes, Mohamadi, Goudarzi, McProud & Hollenbeck, 2006). Food intake was measured using 3-Day self reported dietary records. Each participant was asked to keep a record of food intake over a three-day period, either Wednesday, Thursday and Saturday or Sunday, Monday and Tuesday. Participants were asked to keep track of the portion, type of food including brand if

[1] While there is no law that federally mandates physical education, every state excepting Iowa has state standards for physical education that school districts must follow for grades K-12 (National Association for Sport and Physical Education & American Heart Association. (2012). 2012 Shape of the Nation Report: Status of Physical Education in the USA. Reston, VA: American Alliance for Health, Physical Education, Recreation and Dance.).

possible and the relative amounts of ingredients in combinations of foods like burritos and pizza. A researcher then checked the dietary records for completeness, clarity and accuracy. While this study made efforts to make the self-reported food intake as reliable as possible, there is inherent bias in dietary self-reports. Studies have found that obese people typically underreport their caloric intake (Lichtman, Pisaraska, Berman, Pestone, Dowling, Offenbacher, Weisel & Heshka 1992), which makes this data more unreliable as a specific indicator of caloric intake. While the data on caloric intake should be used as more of an estimate than fact, the outcomes of the study regarding the type of foods ingested still suggests that there is significant dietary change from high school to college, which may be induced by the collegiate environment.

Research suggests that the transition into college is accompanied by a decline in overall diet quality (Nelson, Story, Larson, Sztainer & Lytle, 2008). A longitudinal study by Larson et al. followed students for five years, from high school through their first year of college and tracked their eating behavior. Eating behavior including vegetable and fruit intake, breakfast eating and fast food frequency (Larson, Neumark-Sztainer, Story, Harnack, Eisenberg, & Wall, 2008). The study found that frequency of fast food intake at baseline was inversely related with follow up fruit and vegetable intake. The more fast food students ate, the less likely they were to eat vegetables over the 5 years of tracking. The study also found that socio-environmental factors played a key role in fruit and vegetable intake. Home fruit and vegetable availability and family-meal frequency at baseline were positively associated with the intake of fruit and vegetables (Larson, Neumark-

Sztainer, Story, Harnack, Eisenberg, & Wall, 2008). This finding highlights a problem many college students face. College students frequently live in dorms, apartments or houses (Nelson et al. 2009 report that in the population studied, 75% of students attending college live in dorms or rent apartments or houses) away from their families, making parental support for healthy eating and access to healthy foods more difficult (Nelson, Larson, Barr-Anderson, Neumark-Sztainer, & Story, 2009).

In a separate study, Larson et al found that total fruit and vegetable intake over the same 5 year transitional period from high school to college decreased by more than half a serving a day (Larson, Neumark-Sztainer, Hannan, & Story, (2007). Both Nicole Larson and Melissa Nelson are part of a team of researchers at the University of Minnesota School of Public Health participating in a longitudinal study called "Project EAT (Eating Among Teens)". Their studies look at both eating behavior in high school and maintain the longitudinal study by tracking eating behavior into college. As such, the population base for their studies is primarily Minnesotan youth and young adults. Though this specific population may not generalize perfectly to the US population, the studies are racially and socio-economically diverse and representative of larger population samples.

A study performed by Niemeir et al. (2006) supports Larson et al. and Nelson et al.'s findings that overall diet quality decreases during the transition to young adulthood and the beginning of college. This study used a nationally representative sample provided by the National Longitudinal Study of Adolescent Health, which was analyzed to determine the change in fast food and breakfast consumption over 5 years. The study found marked increases in fast food consumption and decreased

in breakfast consumption occurred over the 5 year interval. High rates of fast food consumption during the initial assessment predicted higher BMI at the subsequent assessment, as did fewer original days or a decrease over time in breakfast consumption (Niemeir, Raynor, Lloyd-Richardson, Rogers, & Wing, 2006).

At the initial assessment, 71.3% of adolescents were classified as normal weight and 28.7% were classified as overweight based on BMI (Niemeir, Raynor, Lloyd-Richardson, Rogers, & Wing, 2006). Of the 28.7 classified as overweight, 10.9% were classified as obese. At the follow up assessment 47% of young adults were classified as overweight with 21.2% of that 47% being classified as obese. This represents a 10.3% increase in obesity in 5 years.

While the collegiate environment may not be the sole contributing factor to increases in weight gain over the 5 year period that includes the transition to college, this study shows that habits such as fast food consumption are associated with weight gain in a college aged population. Fast food consumption has implications for cognitive functioning due to the high dietary fat content typical of most fast foods (Niemeir, Raynor, Lloyd-Richardson, Rogers, & Wing, 2006). Additionally, studies have shown that individuals eat a similar volume of food regardless of the energy density or macronutrient composition of the food being consumed. This implies that an individual may eat past their actual caloric need when consuming fast food, increasing their saturated fat and sugar intake.

WESTERN DIET LEADS TO OBESITY

Researchers have known that the western diet promotes excessive food intake and obesity in rats since the 1980's (Rolls, Rowe & Turner, 1980). Rolls et al

found that adult male hooded rats that where offered a mixed, high energy diet for 90 days became significantly obese compared to chow-fed control rats. Additionally, this study found that when the high energy foods were withdrawn after 90 days, the obese rats maintained their elevated body weights. Half of the experimental group rats were then fasted for 27 days in order to reduce their body weights to control levels. After body weight reached control levels, the half of the experimental rats that were fasted were given unrestricted access to regular chow. These rats then returned to the previously elevated body weight. The study found that eighteen weeks after the initial withdraw of the high-energy western diet, experimental rats had significantly elevated body weights and fat stores.

While it would most likely be unethical to perform a similar experiment with humans, where people were monitored and fed a high energy, western diet over an extended period of time in order to gage the effects of this diet on obesity, studies have correlated the consumption of a western diet with obesity.

Hill et al., evaluated studies by Lissner et al. 1987, Stubbs et al. 1995 and Thomas et al. 1992 that looked at the effect of diet composition on short term (less than two weeks) energy regulation in humans (Hill, Melanson & Wyatt, 2000). The outcomes of these studies showed a surrogate model for the development of obesity, contributing the promotion of obesity to the increase in energy intake that results from these diets or the deregulation in energy or fat balance that results from these diets.

Because these studies took place in a lab setting, researchers were able to record the exact amount of food the participants were offered and the exact amount

ingested. This also allowed researchers to accurately account for the total number of calories ingested. The experimental design is an improvement on the unreliable self-reported data that is commonly used in studies accounting for energy intake because it allows for this accurate measurement. Another benefit of this design is that it allowed researchers to control and record the nutritional composition of the diet.

While there are drawbacks to these studies, like the fact that they are not long term and that these studies compared the effects of diets with 40% or more energy from fat to those containing 20% or less energy from fat (leaving the effects of the range from 20% to 40% energy from fat unexplored), they were able to show consistent results (Hill, Melanson & Wyatt, 2000). The studies showed that humans had a higher energy intake when consuming a high fat diet and that subjects may consume more when provided a diet high in fat. This over consumption was attributed to the higher energy density of the diets (Stubbs et al. 1995), the palatability of the diets (Schiffman et al. 1998) or other behavioral and metabolic differences in the subjects (Hill, Melanson & Wyatt, 2000). The assumption is that higher energy intake contributes to obesity as the increase in available energy is not compensated for in the energy expenditures of the body.

Cross-sectional studies have shown a positive relationship between dietary fat intake and indices of obesity (Lissner & Heitmann, 1995). Lissner and Heitmann compiled data from cross-sectional studies and found that in a significant number of studies there is a positive association between percentage of energy from fat and BMI in populations that consume the western diet. There are many flaws inherent in

these studies including the reliance on self-reported food consumption, which has been shown to be under reported by obese individuals by as much as 30% (Lichtman, Pisaraska, Berman, Pestone, Dowling, Offenbacher, Weisel & Heshka 1992), and the inability of diet surveys to accurately capture the real life variability in food consumption and fat and sugar intake. Despite the drawbacks, the general association of western diet consumption and obesity supports the findings of animal studies and short term human studies in real human models.

THE WESTERN DIET

Diet is central when considering the rise of obesity in the US. The western diet is characterized by a high intake of saturated fatty acids, trans fatty acids and processed foods combined with a low intake of mono-and poly-unsaturated fatty acids, dietary fiber and micronutrients (Neustadt, 2011). In actual food terms, this is typical of foods like grain-fed beef, processed meat (hotdogs and deli meat), refined-grain products, eggs, French fries, high-fat dairy products like cheese, sweets and other deserts. Not only do these foods high in fat and sugar contribute to obesity by increasing energy consumption in an individual's diet, there is also research that indicates that this consumption may corrupt neural functions in brain systems involved in memory and cognitive processing (Berthoud, 2012; Davidson, Sample & Swithers, 2013, in press).

HUMAN STUDIES OF DIET AND OBESITY

Human studies have associated staples of the western diet and increased BMI with a number of negative effects on the brain and cognitive functioning. Many

studies linking the western diet to impairments in cognitive functioning in humans involve middle aged to elderly populations.

Studies of adults have shown that components of the Western diet contribute to cognitive decline. Kalmijin et al. found that among middle aged adults in the Netherlands, the risk of impairment in memory, speed, and flexibility increased by 15% to 19% as saturated fat intake increased (Kalmijn, van Boxtel, Ocké, Verschuren, Kromhout & Launer, 2004). Additionally, higher dietary cholesterol intake was associated with an increased risk of impairment in memory and cognitive flexibility. This study used a cross-sectional approach to look at the effects of saturated fat and cholesterol on cognitive function. A self-administered food-frequency questionnaire was given to each participant in order to assess the habitual consumption of a number of food items and therefore get an idea of overall saturated fat and cholesterol intake. Cognitive function was assessed using a number of neuropsychological tests including the Visual Verbal Learning Test, the Concept Shifting Task, an abbreviated Stroop Test, the Letter Digit Substitution Test and a Category Fluency Test, that tested memory function, speed of cognitive processes and cognitive flexibility (Kalmijn, van Boxtel, Ocké, Verschuren, Kromhout & Launer, 2004).

The study found that the effect of eating 80mg more of cholesterol a day was similar to the effects that being 3 years older would have of cognitive flexibility (Kalmijn, van Boxtel, Ocké, Verschuren, Kromhout & Launer, 2004). The authors do note that their findings indicating cognitive impairment would probably not have any functional significance because the impairments were subtle and the

participants were middle aged. The authors of this study believe that though the pathology of these impairments is subtle at middle age, these impairments are expected to increase with age, leading to Mild Cognitive Impairment (MCI), Alzheimer's Disease or other issues related to dementias. The findings of this study demonstrated that the consumption of components of the western diet are associated with cognitive impairment in middle age and supported suggestions made by other studies that look at the relationship between fatty acids and dementias (Kalmijn, Launer, Ott, Witteman, Hofman & Breteler, 1997).

Eskelinen et al., studied the associations of midlife fat intake to cognitive performance and looked specifically at the occurrence of MCI later in life (Eskelinen, Ngandu, Helkala, Tuomilehto, Nissinen, Soininen & Kivipelto, 2008). The study used a food frequency questionnaire to determine typical saturated fat, polyunsaturated fat and monounsaturated fat intake. The study used the Mini Mental State Examination to assess global cognitive functioning, immediate word recall tests to assess episodic memory, The Category Fluency Test to assess semantic memory, the Purdue Peg Board task and letter digit substitution test to assess psychomotor speed, the Stroop test to assess executive function and a task by Einstein to assess prospective memory (Eskelinen, Ngandu, Helkala, Tuomilehto, Nissinen, Soininen & Kivipelto, 2008).

Einstein's task to assess prospective memory involved two levels of attentional demands outside of the base task. Each participant was show a yellow word on a screen. The participants were told to press the slash key whenever they saw that same word appear again on the screen. The word appeared once in yellow

and three times subsequently in the normal color. For the standard attention condition, participants were asked to monitor the words on the screen for the reappearances and were asked to rate a list of words on various dimensions. In the demanding attention condition the participants monitored the screen for the reappearance of the word, rated words on various dimensions and monitored an audiotape for the occurrence of the number nine (Einstein, Smith, McDaniel & Shaw, 1997).

The study found that individuals who developed MCI later in life were less educated and had higher serum cholesterol levels at midlife (50-51.7 years old) than participants who did not develop MCI later in life (Eskelinen, Ngandu, Helkala, Tuomilehto, Nissinen, Soininen & Kivipelto, 2008). The study also found that high total fat and high saturated fat intake were associated with an increase in risk for MCI. Participants whose total fat intake was high performed significantly worse on tests of global cognitive function and psychomotor speed while saturated fat intake was specifically associated with poorer global cognitive function and prospective memory. Conversely, high intake of polyunsaturated fats was associated with better semantic memory. The study notes that while dietary fat intake is strongly associated with cognitive impairment, the mechanisms by which dietary fats affect cognitive performance are unknown (Eskelinen, Ngandu, Helkala, Tuomilehto, Nissinen, Soininen & Kivipelto, 2008).

Eskelinen et al. provide more evidence that components of the western diet increase the risk of cognitive impairment for middle-aged populations and that subtle signs of cognitive impairment can be seen and tested at middle age.

Unlike adult studies, studies that associate obesity and cognitive function in children generally look at academic performance and performance on cognitive tasks as indicators of cognitive impairment. These studies have not correlated specific components of the western diet with cognitive function. Studies have linked obesity, a consequence of an obesogenic environment, with poor academic and cognitive performance in children.

Datar and Sturm found that students who were classified as "never overweight" during the period from kindergarten to third grade (roughly 5 years to 9 years of age) consistently performed significantly better on reading and math tests than children classified as "always overweight" (overweight for the entire period of the study) or "became overweight" (during the period of the study) (Datar & Sturm, 2006). This study looked at student records from the Early Childhood Longitudinal Study- Kindergarten Class and tracked a group of students from kindergarten to third grade.

Guxens et al. looked at the association between overweight and cognitive function in preschool aged children and found that children scoring lower on cognitive tests at 4 years of age had an increased risk of becoming obese by 6 years of age (Guxens, Mendez, Julvez, Plana, Forns, Basagana, Torrent & Sunyer, 2009). Conversely, higher general cognitive function at four years was associated with reduced odds of becoming overweight at age 6 years. Children that displayed higher abilities in executive function, verbal, quantitative and memory skills were associated with a lower likelihood of being overweight at 6 years of age. Though this seems to suggest that cognitive ability and function may influence the development

of overweight, this conclusion cannot be reached from this study due to the low variability of BMI data at age 4. The low variability in BMI scores at 4 years implies that obesity at 4 years could not be assessed. Future studies may improve upon this research by analyzing the type of food environment the children were exposed to during their first 4 years of life. Specifically, future studies may observe if the children are consuming a typical western diet and look for a relationship between the type of diet consumed and obesity later in life. This study provided further evidence that implies a relationship between cognitive function and weight gain.

Another study performed by Li et al. (2008) used a cross-sectional design to look at the relationship between BMI, academic performance and cognitive functioning in students aged 8-16 that participated in the Third National Health and Nutrition Examination Survey (Li, Dai, Jackson, & Zhang, 2008). This study assessed certain types of cognitive functioning through the block-design and digit-span subtests of the Wechsler Intelligence Scale for Children, Revised and the reading and arithmetic sections of the Wide Range Achievement Test, Revised. The block-design test required participants to use a set of three-dimensional cubes to recreate two-dimensional patterns. The task was used to assess spatial memory, nonverbal reasoning and spatial function. The digit-span test was used to assess attention and working memory and required participants to repeat an increasingly long series of numbers forward and backward. The WRAT arithmetic section evaluated math skills while the WRAT reading section evaluated letter recognition and word-reading skills (Li, Dai, Jackson, & Zhang, 2008).

Using linear regressions, the study found that the association between BMI and cognitive function was statistically significant and that z-scores of tests decreased as the percentile of BMI increased even after adjustments were made for all potential confounding or mediating variables (Li, Dai, Jackson, & Zhang, 2008). After dichotomizing continuous variables, only block design continued to have a "strong and significant" association with body weight. The logistic regression found that the proportion of poor performers tripled among overweight children compared to their normal weight peers on the block design task. The association between obesity and impaired cognitive performance, specifically on the block design task, implies that some negative cognitive effects of obesity and the western diet may be seen as early as childhood.

The difficulty of controlling human participant's diet and the general need to rely on self-reported diet data makes human studies connecting the western diet with cognitive functioning prone to significant and multiple sources of error. Animal models have more directly implicated the western diet in impairing specific cognitive functions. Animal studies provide a reliable way to look at how specific diet components, like saturated fat, regular sugar, and processed sugar, contribute to obesity. Once fed a diet deriving from these components, tests on how the specific substances affect specific cognitive function may be performed.

ANIMAL STUDIES OF THE WESTERN DIET AND COGNITIVE FUNCTION

Animal studies are able to show more direct connections between components of the western diet and impaired cognitive functioning that the previously discussed human studies. The following studies highlight the effects that

high-sugar diets, high-fat diets, and high-fat and high-sugar diets have on different aspects of cognitive function. The impairments that can be seen in these studies include impaired spatial memory and learning and impaired hippocampal functioning. The results of these studies have implications for humans that may provide the basis for future research with human subjects.

Jurdak, Lichtenstein and Kanarek (2008) found that a chronic intake of sucrose solution as a supplement to standard chow was accompanied by deficits in cognitive behavior. In this study, young rats were divided into three different diet groups. One group was given a standard chow diet, one group was given a standard chow diet supplemented with access to partially hydrogenated fat (Crisco) and the final group was given a standard chow diet supplemented with 32% sucrose solution. The rats were fed this diet for five weeks and then exposed to the Morris Water Maze, which is commonly used to assess spatial learning and memory. Spatial learning and memory are believed to be dependent on the hippocampus. An impairment in spatial learning or memory would indicate some type of damage to the hippocampus, which may also control certain eating behaviors and food consumption processes.

Rats that were given access to supplemental sugar or fat consumed significantly more calories than rats fed only chow (Jurdak, Lichenstein & Kanarek, 2008). Sucrose fed rats consumed 56.3% of their total daily caloric intake as sucrose while the rats on the fat supplemented diet consumed 55.3% of their calories as fat. Rats that were given supplemental sugar and fat weighed significantly more than the rats fed only chow at the end of 5 weeks. It is important to note here that this

may not be representative of the western diet, as people exposed to the western diet do not consume 56.3% of their calories from sugar. Instead, this should be viewed as an exaggerated high-sucrose diet resulting from ad libitum access to sucrose solution by rats.

The study found that rats fed the supplemental sucrose diet took significantly more time to complete the Morris Water Maze (MWM) than rats exposed to the other two conditions. Additionally, a probe test implemented 10 days after exposure to the MWM found that sucrose supplemented rats spent significantly less time swimming in the quadrant of the maze where the platform was located than the rats exposed to the other conditions (Jurdak, Lichenstein & Kanarek, 2008), which seems to indicate a lack memory related to the location of the platform.

Though fat supplemented rats performed similarly to the chow only rats, other studies have shown that high fat diets significantly affect cognitive function in an animal model (Molteni, Barnard, Ying, Roberts & Gomez-Pinilla, 2002; Greenwood, & Winocur 1996; Greenwood & Winocur, 2005). A study performed by Molteni et al. found that rats fed a high-fat and sugar diet took more time to find the platform in the MWM than rats fed a low-fat, complex carbohydrate diet. Though this study's results may seem at odds with Jurdak et al. (2008), there were important differences between the two studies that may explain the difference in findings. Moltini et al. (2002) emulated the western diet by giving an experimental group of rats a diet that was high in saturated and monosaturated fat, primarily from lard and a small amount of corn oil, which provided for about 39% energy. The

diet was also high in sucrose, with about 40% energy from sucrose. When compared to the diet in Jurdak et al. (2008) initial differences in diet may be observed.

Jurdak et al.'s rats were given ad libitum access to ground Purina chow and the experimental group's diet were supplemented with either hydrogenated fat (Crisco) or a 32% sucrose solution. The sucrose supplemented group received 56.3% of their total calories from sucrose while the fat supplemented group received 55.3% of their total calories from fat. The main difference in diet is that in Jurdak et al. (2008), high-fat and high-sucrose diets were evaluated separately, while in Moltini et al. (2002) the diet was high in both fat and sucrose.

Additionally, rats in Moltini et al. (2002) were provided a high-fat and sugar diet for 8 weeks while rats in Jurdak et al. (2008) were only provided a high-fat diet for 5 weeks. The duration of exposure to these diets may have influenced the results, leaving the possibility that measurable cognitive deficits could occur after 8 weeks of exposure to the high-fat diet in Jurdak et al. (2008) open for speculation. The type of rat used in the two studies also differed, with Jurdak et al. (2008) using Long-Evans rats and Moltini et al. (2002) using Fisher 344 rats. The stain and breeding of rats may interact with dietary fat in the development of obesity and performance on the MWM (Jurdak, Lichenstein & Kanarek, 2008).

The rats in Jurdak et al. (2008) were also markedly younger than rats typically used for studies, which may help account for why deficits in performance on the MWM in rats fed a high-fat diet were not seen. This study showed that high sugar consumption in rats led to impaired spatial memory and learning (Jurdak, Lichenstein & Kanarek, 2008). While this may not seem relevant to human academic

performance or self-regulation, it indicates a possible impact of long term or excessive sugar intake on the hippocampus. If one type of memory that derives from the hippocampus is impacted by high-sucrose and western diet type consumption, it may be that other aspects of hippocampal function are also impacted.

Recent studies have suggested that the hippocampus may be particularly susceptible to impairment caused by aspects of the western diet (Kanoski, Zhang, Zheng & Davidson, 2010). In order to gain more insight into the western diets effects on cognitive function, Kanoski et al. assessed the degree to which the consumption of a high-energy diet consisting of lard-based saturated fat and glucose affected specific hippocampal functioning. This study used hippocampal dependent tasks to test cognitive function instead of more general tasks like the Stroop test that measure executive function or more general global cognitive functioning. The study found that consumption of a western-like diet consisting of high-fat and high-sugar impaired performance in hippocampal dependent discrimination problems but not in discrimination problems that do not rely on the hippocampus.

Kanoski et al. (2010) used nonspatial Pavlovian discrimination problems to assess hippocampal function. These problems included a serial feature negative discrimination task, a feature positive discrimination task and a non-conditioned discrimination task (Kanoski, Zhang, Zheng , & Davidson, 2010). Holland et al. showed that rats with hippocampal lesions were impaired in learning a feature negative discrimination though these effects were not seen in feature positive and non-conditional discriminations (Holland, Lamoureux, Han and Gallagher, 1999). If rats show impaired ability to perform a feature negative discrimination task, but are

not impaired in performing a feature positive or non-conditional discrimination, this would imply that similar regions of the hippocampus are effected by the western diet.

The rats were separated into groups and received feature negative training sessions that involved a feature negative discrimination and a non-conditional discrimination or feature positive training sessions that involved a feature positive discrimination and a non-conditional discrimination. The study found that rats fed a high-energy, western diet exhibited impaired serial negative discrimination performance compared to rats fed a standard chow diet. Rats that were fed the high energy, western diet were not impaired in solving feature positive discriminations when compared to rats fed a standard chow diet and the high energy diet was not associated with impaired non-conditional discrimination learning in either feature negative or feature positive discriminations. Additionally, the consumption of the high energy diet produced significant increases in body weight for rats during the 90 day ad libitum period while standard chow diet consumption did not produce significant weight gain (Kanoski, Zhang, Zheng , & Davidson, 2010).

These results show that in an animal model, learning and memory processes that rely on the integrity of the hippocampus are more susceptible than other processes to impairment caused by the western diet (Kanoski, Zhang, Zheng, & Davidson, 2010). Research by Davidson et al. (2009) has shown that rats with selective hippocampal lesions display elevated food intake and body weight gain in comparison to non-lesioned controls. This may indicate that the western diet,

through impairing hippocampal function, also promotes excessive energy intake and over eating that leads to obesity.

HUMAN STUDIES OF DIET AND COGNITIVE FUNCTION

In the past decade, studies have connected obesity with an increased risk for Alzheimer's disease (Solfrizzi, 2002). More specifically, studies have linked consumption of saturated fats and simple carbohydrates to the development of cognitive dementias (Kanoski & Davidson, 2011). Cognitive dementias include Alzheimer's Disease and Mild Cognitive Impairment (MCI) which is a "diagnosis given to individuals that exhibit deficits in memory, language or other mental functions that exceed what is expected as part of normal aging, but that do not interfere significantly with their daily activities" (Peterson et al, 1999). Animal studies have shown that the western diet contributes to deficits in learning and memory, specifically in relation to the hippocampus.

Abnormalities in the hippocampus are associated with the early stages of Alzheimer's disease and other cognitive dementias (Mungas, Jagust, Reed, Kramer, Weiner, Schuff et al., 2001). David Smith used imaging to track the progression of Alzheimer pathology through the human brain and found that deficits in verbal memory correlate with atrophy of the left hippocampus and deficits in nonverbal memory correlate with atrophy of the right hippocampus (Smith, 2002). Smith also found that a small proportion of neuron tangles, which appear in abundance in late stages of Alzheimer's and are believed to contribute to the disease, can be seen through imaging 43 years before mild cognitive impairment occurs (Smith, 2002). The early identification of Alzheimer's disease pathology is supported by the

findings of Elias et al. (2002), who found that minor deficits in learning and retention and abstract reasoning begin to appear up to 22 years before the development of Alzheimer's disease

With the implication that western diets contribute to Alzheimer's disease, and with the ability to identify early indications of Alzheimer's disease, it is surprising that there is so little research available on the effects of the western diet on the cognitive function of a college aged population. Even if there is no evidence of Alzheimer's disease pathology in a college aged population, or even precursors to Alzheimer's disease, over consumption of the western diet and obesity may still lead to cognitive impairment, which could become more serious later in life. Because of this, further research into the western diet, cognitive impairment and a college-aged population is necessary.

Evidence provided in many studies suggests that the western diet leads to cognitive impairment in adults (Smith, Hay, Campbell & Trollor, 2011). High intake of saturated fats and sugar increases a person's risk for developing a disorder on the dementia spectrum later in life (Whitmer, Gustafson, Barrett-Connor, Haan, Gunderson & Yaffe, 2008). Not only are these effects seen later in life, studies have shown that effects can be felt in midlife and may be able to be identified, through academic performance, as early as childhood (Kalmijn, van Boxtel, Ocké, Verschuren, Kromhout, & Launer, 2004; Li, Dai, Jackson, & Zhang, 2008). A link has been drawn between obesity and poor cognitive performance in children, providing support for the idea that cognitive function is significantly affected by components of the western diet early in life and has negative effects on cognition throughout a

person's lifetime (Li, Dai, Jackson, & Zhang, 2008). If the effects of the western diet on cognitive functioning may be seen in terms of performance as early as childhood, it is logical that signs of cognitive impairment might also be seen in a college-aged population.

Future Research in a College Aged Population

The research on cognitive impairment and the western diet is extremely limited for the 18-24 year old (college aged) range. There are only a handful of studies looking at obesity and cognitive impairment, much less analyzing the possible components of the western diet that might be responsible for any cognitive decline found. David Smith provides images of the progression of Alzheimer pathology through the brain, indicating that the start of this pathology begins within a person's 20's (Smith, 2002). While there are no indicated symptoms of Alzheimer's disease at this stage of pathology (Smith, 2002), the start of pathology implies that there is some type of degradation occurring in the brain at that point in time.

Further research on the possible cognitive impairments in a college-aged population may show that signs of degradation or general cognitive impairment can be seen in a college aged population. Based on studies linking diet, obesity and cognitive impairment in both adult populations and children, I would expect that if tested, obese and overweight college- aged populations would show preliminary signs of cognitive impairment. This impairment would most likely relate to memory function and inability to regulate energy intake. It is also possible that deficits in executive functions and psychomotor speed may be observed. Research into this critical period of weight gain, diet change and initial pathology could provide an

opportunity for early intervention and could help to decrease the instances or severity of Mild Cognitive Impairment, Alzheimer's and Dementia later in life.

FURTHER RESEARCH

In order to gain more insight into possible cognitive impairment in a college-aged population, students eating habits, BMI and cognitive functioning should be tracked to monitor a possible relationship between an increase in western diet consumption and a decline in cognitive function. Ideally, this would be completed by obtaining a representative sample of college aged volunteers to participate in a longitudinal study throughout college, with follow up evaluations five, ten, thirty and fifty years after the initial four year monitoring period. Though this would be ideal, realistically thirty and fifty year follow-ups are impractical and difficult. While it is important to assess the possible long-term effects of the western diet, which could be assessed 50 or 60 years after the initial four-year period, continuing to engage people in a study for that period of time would pose daunting problems, primarily regarding drop out rates. Because of this, this study will perform an initial four-year assessment and follow-up ten years after the four-year assessment.

There are inherent difficulties in maintaining a longitudinal study that may become a factor in this study. In any longitudinal study there is a likelihood that of some participants will drop out due to a physical change in location or general inability or desire to complete the study. In this study there is also the possibility that participants will transfer universities or drop out of school during the initial four-year monitoring period. In long-term follow up sessions, mortality rates may affect the number of participants in the study. Mortality may affect the obese

population in the study disproportionally as obese people are more likely to have health problems that lead to an earlier death than their normal weight peers. Demented subjects may also contribute to the mortality rate in the study, as the drop out rate for both the obese and demented subject will likely be higher across any long-term longitudinal study.

The goal of this study would be to correlate western diet consumption with impaired cognitive functioning in college-aged adults. Specifically, impairments involving hippocampal functions that may impact academic success and promote excess energy intake and weight gain would be of interest. Studies have found that the western diet impairs the hippocampus in ways that are similar to the effects of selective hippocampal lesions, impacting memory and the ability to control energy intake and regulation in animal models (Kanoski, Zhang, Zheng & Davidson, 2010).

When memory retrieval is viewed as a progression from retrieval cues to items stored in memory by way of associative links, the ability to inhibit memories is integral to retrieving the target response. When a retrieval cue is associated with more than one memory item, non-target memories that are associated with the same retrieval cue "compete" with the target memory for conscious recognition (Anderson & Neeley, 1996). The ability for a person to recall the correct memory then hinges on the persons ability to inhibit the incorrect, or non-target memories that are competing for access to consciousness. Proposed models indicate that the impaired ability to inhibit or control memories of positive digestive outcomes of palatable food intake may lead to greater food intake and an increased inability to self-regulate consumption.

Executive cognitive function is considered an umbrella term for cognitive processes that regulate, control and manage other cognitive processes (Ellicott, 2003). Executive cognitive function encompasses a number of interrelated subskills including the ability to inhibit strong impulses and emotions, to shift attention from one task to another, to plan, to initiate tasks and to utilize working memory (Pennington & Ozonoff, 1996). As such, executive function and memory inhibition may be interrelated as both involve hippocampal activation. This study will utilize tasks that measure both memory inhibition and executive function because of this possible relationship.

The increased consumption of unhealthy foods typical of a western diet may contribute to greater BMI, increase hippocampal impairment and lead to continued difficulty with self-regulation of diet for the obese. The inability to inhibit competing memories to recall the correct memory may also affect academic performance. When attempting to recall a specific fact or memory for an academic test (the target memory), the retrieval cue, or test question, may be associated with many memories that deal with the same topic being tested. In order to succeed on the test, the non-target memories must be inhibited so the target memory may be accessed by consciousness. If the ability to inhibit competing, non-target memories is impaired and a non-target memory is allowed into consciousness and recorded on the test, this test answer would be incorrect thereby lowing the test takers grade as a measure of academic performance.

Because the college years have been identified as high-risk years for weight gain, studying a college population provides interesting and unexplored

opportunities. These opportunities include monitoring possible change in cognitive functioning as students gain weight as opposed to after an individual is already overweight or obese (this may provide a more direct way make a correlation between students gaining weight and decreasing cognitive function). Looking at a younger population for potential signs of cognitive decline also creates an opportunity for intervention initiatives. Following up with participants five, ten, thirty and fifty years after completing the initial study may allow connections to be draw between diet at young adulthood, outcomes in middle age and eventually the development (or lack thereof) of mild cognitive impairment, Alzheimer's or other diseases on the dementia spectrum. Though this study will only focus on an initial four-year evaluation and a ten-year follow-up, this study may provide the foundation for other researchers to build off of and would provide additional data for other researchers who are looking at representative samples of the participants of this study in an older population.

In this study, participants would be recruited at the beginning of their freshman year of college. At this point, BMI would be measured, basic health information would be solicited (i.e. family medical history, personal medical history, diabetes, cardiovascular disease etc.), demographic information would be taken, current medication use, blood pressure would be taken, adiposity measured and a self-reported food frequency questionnaire would be administered. The food frequency survey would be used to get a rough idea of the participants diet prior to contact with researchers. After the initial food frequency survey is administered, participants would be asked to track their food intake for a week prior to each

session with researchers. This week measure is used in order to get a general idea of caloric intake during a typical week for the participants. A week measure will be more accurate than administering a food frequency survey for the past four months every time participants meet with researchers. The relatively short duration of the required tracking (a week) is feasible for participants without being burdensome, and this will also allow researchers to look at a typical weeks caloric intake. While still not entirely reliable, having participants track for the week before their session with researchers would allow researchers to generalize the diet of the participants over a semester.

Participants will be selected from the incoming freshman class at a participating university. Participants must be between 17 and 19 years of age, a freshman at the university, enrolled full time and accepted into a 4-year degree program. Participants would also have to agree to allow researchers to access academic records and any other indicators of academic performance. Of those students that meet the criteria, a random sample will be taken to get a total of 76 student participants. The goal of the study is to begin with 76 participants and ideally maintain all 76 participants throughout the longitudinal study. In order to encourage participation, appropriate incentives for participation may be offered. These incentives may include credit towards required study participation hours present in some university classes or small monetary incentives. These participants should be representative of a larger population in terms of race, ethnicity, body weight and adiposity. The study should be as equal as possible in terms of male to female ratio.

Participants would meet with researchers at the beginning of their first semester of college and at the beginning of each subsequent semester. While not perfectly even, this provides roughly the same amount of time between sessions when operating on a two semester system (classes beginning in August and ending in May, with an extended winter break). At each session, BMI, waist circumference and adiposity will be measured, changes in medication and health status will be recorded, blood pressure will be taken, and academic performance will be assessed.

Drop out rate is a concern for this study. Students who are performing poorly academically may be forced to drop out due a need to focus more time and energy on school or because they can no longer attend university. Other students may have to leave school due to a change in financial situation. Students who are performing poorly would be of interest to this study, as one of the premises underlying the study is that the western diet may cause cognitive impairment that affects academic performance. An exit survey will be administered to students who drop out in order to determine the participant's reasons for dropping out. Depending on the cause of leaving (i.e. financial reasons would not be grouped in the same category as poor academic performance) these participants would be compared during analysis to students still in the study and therefore still attending school, in order to determine any differences in western diet intake or cognitive performance on the administered tasks. An attempt would also be made to follow up with students who drop out of the study during the ten-year follow-up.

In order to measure cognitive function in relation to memory inhibition, a Think/No Think task will be used. This task will be varied in order to prevent

testing bias, a bias that occurs as the same tests are taken over a period of time. Eventually, people will get better at the tests if they are exposed to the same tests every four months. For example, Anderson and Green's (2001) Think/No-Think paradigm may be used with face-picture pairs or with word pairs (ex: fruit/orange).

In the face pair Think/No Think task, forty female faces that have been normalized for neutral expression were chosen. These faces would then be selected by two independent raters to ensure that the faces were not related, in order to avoid grouping effects. The procedure would be divided into three parts, training, experimental and testing. During training participants would learn the 40 face picture pairs by first viewing all the pairs, and then being tested on the pairs by having one face appear on a screen for four seconds and having to match it with its original pair. The training phase would continue until each participant could identify the right face pair 97.5% of the time.

In the experimental phase, participants will see a face for 32 face pairs instead of the total 40. Sixteen face pairs will be used in the think condition and 16 will be used in the no-think condition. Each condition will be indicated by a colored boarder around the face picture, green for think trials, red for no-think trials. Participants will be told to "Think of a picture previously associated with the face" in the think condition and participants will be told "Do not let the previously associated picture come into consciousness". The faces would be displayed on the screen for 3.5 seconds each. For each condition, participants should view the randomly distributed faces 12 times. During the test phase, participants will be show each of the phases and asked to write down a description of the originally

associated face. This will help provide accuracy measures for the rest of the study (Depue, Burgess, Willcutt, Ruzic & Banich, 2010). The Think-No Think tasks, whether used with face pairs or word pairs measures the ability of a person to inhibit memories.

In the word pair Think/No Think task, participants will be asked to learn a list of unrelated word pairs so that when prompted with one half of the pair they could respond with the other half of the pair. After the training phase, the experimental think/no think phase would have a cue from each pair appear on a screen in either red or green. When the cue word is green, participants would be instructed to think about the associated word and say the associated word out loud. When the cue word is red, participants would be instructed to look at the cue word but not think about or say the appropriate word. A final testing phase would ask the participants to remember the words pairs as best as they could when prompted by a cue word. A good performance on the think condition of the Think/ No Think task for either the face design or the word pair design would indicate an ability to inhibit competing memories in order to answer with the correct, target response. Additionally, a better memory for the No Think words or faces in the sample would indicate that these memories were not inhibited despite the directions in the study designed to help inhibit No Think words and faces.

In order to measure executive cognitive function the Trail Making Test (TMT) will be used. The TMT has two parts, part A and part B. In part A, participants are asked to use a pencil to connect a series of 25 encircled numbers in numerical order as fast as they can. In part B participants are asked to connect 25 encircled numbers

and letters in numerical and alphabetical order starting with numerals and alternating between numbers a letters. The numbers and letters in both part A and part B are places in a semi-random fixed order in a manner that allows no crossing liens to be drawn by the participant. This test is timed, with 300 s being the general cut off and maximum score. Part A is meant to test visual search and motor speed skills while part B is meant to test higher level cognitive skills like mental flexibility. This task is also at risk for testing bias, with participants getting better at the test the more times they are exposed to the test (Bowie & Harvey, 2006). The data and analysis resulting from this research would add to the existing dearth of knowledge about the relationship between diet, obesity and cognition and young adults.

The links between the consumption of a western diet, obesity and cognitive function have been identified through both animal and human studies. This relationship has been shown in both middle-age adult populations and child-age populations, though it has not yet been established in a college-aged population. Though the impact of the western diet on young adults has not yet been addressed, it is important to identify the environment and underlying mechanisms that increase obesity in a college population during this critical period. These underlying mechanisms may negatively impact academic performance and promote continued obesity throughout a lifetime. This obesity, in turn, could cause major health problems including type 2 diabetes, hypertension, various cancers, coronary artery disease and stroke (Kirk, Penney & McHugh, 2009). These chronic ailments could exacerbate existing cognitive impairment and generally heighten the risk for developing Mild Cognitive Impairment and Alzheimer's disease. By gaining more

information on the underlying mechanisms that disrupt cognitive function and promote obesity, better and earlier intervention methods may be developed.

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